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Paper/Poster-Abstract Form

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Title: Responses of lung cell cultures after realistic exposure to primary and aged carbonaceous aerosols

Abstract: (min. 300 – max. 500 words)

The abstracts for papers and posters must contain unpublished information on your research subject: background, investigation methods, results and conclusions. Graphs and references are very welcome. Acronyms should be avoided. Abstracts with < 300 words can not be considered. General information on products which are already commercially available can not be accepted as presentations for the conference but are very welcome at the exhibition of particle filter systems and nanoparticle measurement instruments.

Epidemiology has provided consistent evidence for the link between adverse health effects and increased concentrations of ambient (ultra)fine particles. There is an urgent need for biological experiments aimed directly at the cause-effect relationship. A major fraction of ambient aerosol particles is composed of organic material, whereby oxidizing organic compounds like peroxides in such particles, are possibly responsible for the observed health effects.
We examined the responses of lung cells to primary and photochemically aged aerosols originating from diesel exhaust and wood combustion processed and aged in a large-scale smog chamber. Particles were deposited on cells under realistic ambient air and physiological conditions in a recently developed particle deposition chamber (Savi et al., 2008). Cell cultures, representing the inner surface of airways and alveoli, were microdissected porcine and re-differentiated human airway epithelia with established air liquid interface (ALI), porcine lung surface macrophages, and the human bronchial epithelial cell line BEAS-2B. Cells were exposed to the aerosols at ALI conditions for 2 hours; controls were left untreated in the incubator or exposed to particle free air. Cellular responses were measured within 24 hours after aerosol exposure. Biological endpoint measurements included cell and tissue integrity, phagocytic activity of macrophages, cytotoxicity, release of inflammatory mediators such as interleukin-1β (IL-1β), IL-6, IL-8, tumor necrosis factor alpha and monocyte chemotactic protein-1.
Our data indicate that a single, realistic exposure of lung cells to primary and aged aerosols from diesel exhaust and wood combustion induces mild but distinct cellular responses, providing consistent evidence for potential induction or aggravation of adverse health effects. There is evidence for (i) a decrease in IL-6 and IL-8 release by the airway epithelium after exposure to primary and aged particles originating from both sources, (ii) a potential decrease in phagocytic activity of lung surface macrophages, (iii) similar effects of primary and aged particles from the same source, (iv) similar, but weaker effects of wood combustion aerosol particles compared to particles from diesel exhaust, (v) no effects of the particles on cellular integrity and ultrastructure, and (vi) no major cytotoxic effects.

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References:

Savi, M., Kalberer, M., Lang, D., Ryser, M., Fierz, M., Gaschen, A., Rička, J., Geiser, M. (2008) *Environ. Sci. Techn. 42*, 5667-5674.

Short CV:

L. Künzi: PhD student at the Institute of Anatomy, University of Bern, expected to finish studies in June 2012

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